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HEART RATE VARIABILITY AND THE SUBSTRATE FOR VENTRICULAR TACHYCARDIA IN ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY

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Background: Ventricular tachycardia (VT) in pts with arrhythmogenic right ventricular cardiomyopathy (ARVC) is heart rate and catecholamine dependent. Heart rate variability (HRV) analysis provides a useful method to assess cardiac autonomic function. The aim of this study was to evaluate the relationship between HRV and the substrate for VT in ARVC.

Methods: 16 pts with biopsy-proven ARVC (age 50±13 years) underwent an extensive baseline evaluation, including ECG, SAECG, 24-hour ECG to assess HRV, cardiac MRI, electrophysiologic study with programmed ventricular stimulation, and high-density electroanatomic mapping. Standard definitions of electroanatomic scar, and fractionated, isolated, and very late potentials were used. All patients received an ICD for primary prevention of sudden cardiac death.

Results: During 18±9 months of follow-up, 6 (38%) patients received appropriate ICD shock for rapid sustained VT. No baseline clinical and non-invasive finding predicted the occurrence of ICD shock at follow-up. Electroanatomic scar was present in 14 (88%) patients, and no significant correlation was found between the presence and extension of RV scar the occurrence of ICD shocks at follow-up. Ten (63%) patients presented slow conduction channels (i.e., fractionated, isolated and very late potentials) within scar. Reduced HRV parameters were found in patients with slow conduction channels within the scar (standard deviation of RR intervals [SDNN] 87.8±35.6 vs. 140.2±33.5 ms, P=0.01 in the time-domain; low frequency amplitude [LF] 19.2±15.2 vs. 34.5±13.5 ms, P=0.04 in the frequency-domain). Presence of slow-conduction channels within scar (P=0.034) and reduced HRV parameters (P=0.007 for the SDNN; P<0.001 for the LF) were the only predictors of ICD shock at follow-up.

Conclusions: In patients with ARVC, reduced HRV is associated with fractionated and delayed electrograms within the scar and a higher incidence of VT at follow-up. These findings provide mechanistic insights into the relationship between cardiac autonomic modulation and the arrhythmogenic substrate of ARVC, and suggest that HRV analysis might be useful for the arrhythmic risk stratification of these patients.